

Asbestos in Drinking Water: A Canadian View

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For several years now, public health professionals have been faced with evaluating the potential hazards associated with the ingestion of asbestos in food and drinking water. In Canada, this is a subject of particular concern, because of the widespread occurrence of chrysotile asbestos in drinking water supplies.

The results of available Canadian monitoring and epidemiologic studies of asbestos in drinking water are reviewed and discussed in light of other published work. It is concluded that the risk to health associated with the ingestion of asbestos, at the levels found in municipal drinking water supplies, is so small that it cannot be detected by currently available epidemiologic techniques.

Asbestos is a commercially important mineral in Canada, which is second only to the Soviet Union in world production. Although the mineral also occurs in Newfoundland, British Columbia, Ontario, and the Yukon, mining is concentrated chiefly in Quebec. Asbestos was first mined in Canada in 1878, and production today is about 1,500,000 metric tons/yr (1). About 95% of the asbestos produced in Canada is exported, largely to the United States and Europe.

The hazards to health that can result from inhalation of asbestos dust in the occupational environment have long been recognized, and, as a result, most countries today have an occupational standard of 2 fibers/mL or better. However, asbestos is also a ubiquitous environmental contaminant because of its extensive industrial use and the dissemination of fibers from natural sources. Asbestos has been detected in the air of many of our cities and in our food and drinking water. Therefore, for several years we have been faced with the question of whether the ingestion of asbestos presents a public health hazard. In this workshop we have been fortunate to learn about the results of some very interesting research carried out by U.S. and other scientists in an attempt to answer this question. I would like to review some of the Canadian contributions to this field.

The presence of asbestos fibers in drinking wa-

ter was first reported in 1971 by Cunningham and Pontefract in a study conducted at Health and Welfare Canada (2). In addition to determining the asbestos content of a variety of alcoholic beverages, Cunningham and Pontefract analyzed the drinking water supplies of eight municipalities. Although their methodology could be criticized when compared with today's techniques, these investigators nevertheless provided the first information on asbestos concentrations in public drinking water supplies.

Six years later, in 1977, Wigle (also from Health and Welfare Canada) studied cancer mortality for the period 1964-73 in 22 municipalities in Quebec (3). Two of the municipalities, Asbestos and Thetford Mines, were known to have high concentrations of asbestos in their drinking water supplies. In the Cunningham and Pontefract study, the levels measured were approximately 200 million fibers/L. Asbestos has been mined in these two communities since 1878 and 1882, respectively, so it can be assumed that the population has ingested drinking water containing substantial concentrations of asbestos for many years. Other communities were selected as likely to have high asbestos concentrations in their water supplies because either they were located immediately adjacent to asbestos-containing deposits or they obtained their drinking water supplies from rivers that drain the regions of such deposits. Community water supplies with probable low concentrations were selected, based on their location being remote from peridotite deposits.

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Cancer mortality data and population data were obtained from the Health Division of Statistics Canada. Significantly high cancer mortality for all cancer sites combined was observed for men in both Asbestos and Thetford Mines (observed = 195, expected = 158.4), but not for women (observed = 127, expected = 138.2). Mortality due to cancer in all gastrointestinal sites was not elevated in men or women in these two high-exposure communities. A slight excess of mortality from stomach cancer was observed in men only; similarly, a small excess in mortality from cancer of the pancreas was observed in women only. However, substantial excess lung cancer mortality was noted among men (observed = 78, expected = 46.8). Since a large proportion of the labor force in both Thetford Mines and Asbestos is employed in asbestos mining and milling, it seemed likely that the occupational exposures would account for the excess mortality among males due to cancers of the stomach and lungs. The authors therefore concluded that the study "did not reveal excess cancer mortality that could be related to the presence of asbestos fibres in drinking water supplies."

In 1977, a national survey of asbestos concentrations in drinking water was conducted in Canada (4). Water samples were taken from 71 locations across the country, and the water supplies of more than half of the population were sampled. Most of the sampling was conducted in large cities, but the water supplies of small towns in the mining areas were also examined. Samples of raw, treated, and distributed water were collected from all locations. This type of sampling scheme was used to evaluate the impact of the water treatment and distribution system on fiber concentration.

The samples were analyzed for asbestos content using the U.S. EPA interim procedure. This involved filtration through a Nuclepore filter, which was then carbon-coated under vacuum. The filter material was then dissolved away, leaving a carbon replica of the filter surface with the particles embedded in it. To obtain a good representation of the deposit over the whole area of the filter, three grids were prepared from different parts of each filter. The fibers present in 10 grid openings selected from all specimen grids were counted on the transmission electron microscope. By using this procedure, a precision of $\pm 30\%$ was obtained in most cases, the precision of the fiber count being limited largely by the counting statistics. Morphology was used as the primary criterion for identifying fibers as either amphibole or chrysotile. Supporting evidence was provided by se-

lected area electron diffraction and energy dispersive X-ray analysis.

The survey revealed that amphibole asbestos is not a significant contaminant of Canadian drinking water supplies. Only 7% of 336 samples showed detectable levels of amphibole fibers, and they were generally associated with high chrysotile levels.

Chrysotile asbestos was identified as the major asbestos type present in drinking water. At the time of the survey, 5% of the population received water with asbestos concentrations greater than 10 million fibers/L. About 0.6% received water with more than 100 million fibers/L. The highest result for a tapwater sample in this survey was 1,800 million fibers/L, which was recorded for Baie Verte in Newfoundland. In this particular case, the provincial government initiated measures to reduce the levels. The authors also obtained considerable information about the nature of the fibers in these samples. In general, the median lengths observed were between 0.5 and 0.8 μm . There was also an indication in at least one and perhaps two cities that erosion of asbestos-cement pipe was contributing to the asbestos content of drinking water.

The relationship between the mortality rates for a variety of diseases and the asbestos levels in the drinking water of the surveyed communities was then examined (1). The 71 cities surveyed were divided into two groups: those with asbestos concentrations in drinking water greater than 100 million fibers/L and those with less than 5 million fibers/L. Those cities with intermediate concentrations and those with small populations (less than 10,000) were excluded from the study. There were two cities (Sherbrooke and Thetford Mines) in the relatively high group and 52 cities in the low group.

The death rates from various causes in these two groups were compared in the following way. The localities were defined according to the 1976 census geographic boundaries. All deaths among usual residents of the localities were retrieved from the computerized national mortality files. The population at risk was estimated, using data from the censuses of 1966, 1971, and 1976, and adjusted to 1976 geographic boundaries. Person-years at risk by sex and age during the period from 1966 to 1976 were calculated for each locality. Age-standardized mortality rates (ASMRs) were calculated for the age range of 25 to 69. This range was selected for comparison because the reliability of the certified cause of death in this range is greater than that for older persons. The results showed that the all-cause and all-cancer

mortality rates for males in Thetford Mines were significantly higher than those for Sherbrooke or the 52 comparison localities, but the rates for females were similar in all three regions. Mortality rates for nonneoplastic diseases of the respiratory system were significantly high for males in Sherbrooke and Thetford Mines; the rate for females in Thetford Mines was significantly low.

Particular attention was paid to the incidence of cancers of the gastrointestinal (GI) tract. The only high ASMR of statistical significance among the GI cancers was that for stomach cancer in males in Thetford Mines; the mortality rate of 38.7 was more than double the rate in the comparison group, which was 16.8. No significant difference was observed for other GI cancers between the "high" and "low" groups. The only other statistically significant result was the high mortality rate for lung cancer among males in Thetford Mines.

The authors concluded that, among males in Thetford Mines, the high mortality rates due to lung cancer and stomach cancer were probably caused by occupational exposure. Thetford Mines is one of the oldest asbestos mining communities in Canada.

In this context, it is particularly interesting to examine in detail the data for Sherbrooke. For many years, the source of the drinking water supply for Sherbrooke has been a lake that is contaminated by natural serpentine deposits. The water is not filtered during treatment. In the 1977 survey, the concentration of asbestos in drinking water samples taken from the distribution system was found to be 153 million fibers/L. The population is relatively large (93,000 in 1971), and, most importantly, there is no asbestos mining activity in the immediate region. Age-adjusted mortality rates for Sherbrooke were, therefore, compared with those for seven municipalities with low asbestos concentrations in their drinking water supplies. These municipalities were matched for water source, chlorination status, and population size (50,000–100,000). For both men and women, mortality rates for all GI cancers combined were, in fact, found to be lower in Sherbrooke than in comparison cities. Only minor, statistically insignificant differences were observed for cancers of specific GI sites and indeed, for all other sites.

The Canadian studies thus provide no consistent, convincing evidence of increased cancer risks attributable to the ingestion of drinking water contaminated by asbestos, even though the observed asbestos concentrations were relatively high in several communities.

Although several ecologic epidemiologic studies have been conducted to date, evidence of increased cancer incidence has been associated with the ingestion of asbestos in drinking water in only one investigation, i.e., the one conducted in the San Francisco Bay Area (6). Although this study was carefully designed, several factors complicate the interpretation of the results of the San Francisco Bay study and indeed of ecologic epidemiologic studies generally. Several of these confounding factors have been described in this workshop. It is reassuring that the association observed in the California study has not been confirmed in the Puget Sound case-control type study, which included assessment of exposures and outcomes for individuals rather than populations.

Based on a review of the toxicologic studies on ingested asbestos, we believe that there is no conclusive evidence that ingested asbestos is carcinogenic or cocarcinogenic in animal species. The early studies were inconclusive due to shortcomings in study design. In the more complete studies conducted more recently, the incidence of GI tumors in the asbestos-fed animals has not been significantly greater than that in the control groups. These results were also confirmed by the extensive National Toxicology Program study described at this workshop. There is also no evidence of a dose-response relationship in any of the studies.

The absence of definitive evidence of carcinogenicity in the epidemiologic and toxicologic studies may be due to GI-induced changes that reduce the carcinogenic potential of the fibers. Another possibility is that the GI mucosa presents an effective barrier to the penetration of asbestos fibers. Data concerning the penetration of asbestos fibers into and through the GI tract are contradictory, and the interpretation of the results of available studies is complicated by factors such as the insensitivity of available analytical techniques and the possibility of contamination during sample preparation. It seems unlikely, however, that there is widespread penetration of ingested asbestos fibers into and through the GI tract.

On the basis of the available data, we conclude that the risk of disease associated with the ingestion of asbestos at levels found in drinking water supplies is probably extremely small.

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